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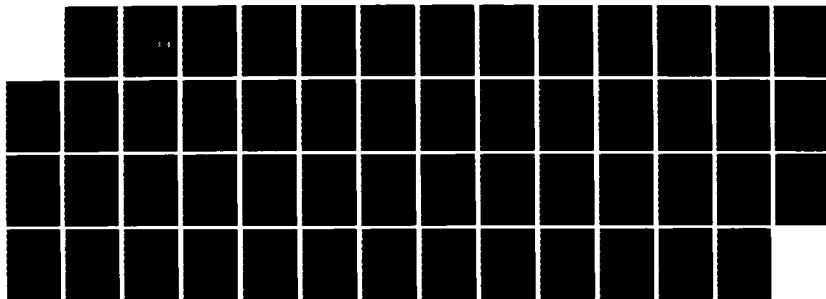
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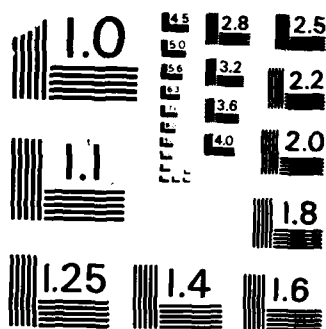
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**Modeling and Simulation II:
Specificity Models for Visual Cortex Development.**

key words: modeling, simulation, visual system, plasticity, synaptic modification, receptive field selectivity.

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Abstract

Observations of developmental plasticity in the visual cortex of cats have inspired a number of theoretical models. These models attempt to mimic the development of response specificity. We review the assumptions upon which one can build models of the visual cortex.

The anatomical layout usually consists of excitatory afference from the lateral geniculate nucleus, with intracortical inhibition among a population of cells. The degrees of convergence of the afference and divergence of the inhibition are some of the more important considerations which distinguish different models. Synaptic modification rules lie at the heart of these theories. Modification is usually of the Hebbian type, with synaptic strengths changing as some function of the product of pre- and post-synaptic activity. A successful model not only alters synapses to generate specificity in single units, but simultaneously produces a cortical network which is stable and which mimics the behavior of experimental populations.

Introduction

In this second of a series of papers, we provide the modeling details of a study in neural development: accounting for the organization of *orientation selectivity* and *ocular dominance* in neurons of kitten visual cortex. In the first paper²⁶ we offered a general introduction to modeling, including a guide to simulation techniques and error analysis, and a review of CAE software packages adaptable for neural modeling.

By *visual cortex* (VC), we mean kitten primary visual cortex, area 17 or V1. We do not consider extra-striate cortex here, or even differences between cat and primate. We first review some pertinent features of *receptive field development*, and then review area 17 models which use the anatomical layout to account for RF features, incorporating synaptic excitation and inhibition. Then we show how to model synaptic *modification*, necessary to explain plastic changes during development.

1. Visual response specificity in kitten and adult cat neurons.

Researchers commonly illustrate *specificity* or *selectivity* by the abstraction of a *tuning curve*, in which the response is small outside of a narrow range of preferred stimuli. In the cat, VC specificity is found over the dimensions of

- receptive field (RF) position,
- stimulus orientation,
- spatial frequency,
- length
- stimulus velocity,

- ocular dominance.
- binocular disparity.

As a particularly good example of specificity, we show in Figure 1 a *disparity tuning curve* from a cat VC cell.

There are several recent reviews of kitten visual cortex development^{24,39,68,99} which the interested reader may consult for details beyond the information presented here.

Since we are interested in modeling development of responses in kittens, we must ask: How do visually naive kittens and adult cats differ in VC specificity? Although this is an area of active research, and there are a number of controversies about the *degree* of difference, it can be said with some conviction that kitten neurons lack much of the adult selectivity. To take the example of *orientation selectivity*, a number of quantitative studies have shown that only a low percentage of cells sampled in kittens have the selectivity for orientation seen in a majority of adult neurons^{1,11,14,15,37,88,98}. Many cells in kitten VC respond *aspecifically* to all stimulus orientations and have large RF's with poorly defined boundaries. The responses may be sluggish -- with few spikes per presentation and fatigue after repeated stimuli. With regard to *ocular dominance* (OD) immature cells usually respond well to stimuli through either eye, and often show mild *facilitation* when tested simultaneously through both eyes. Selectivity for *binocular disparity* is not evident in immature neurons⁸⁸.

In contrast, VC cells recorded in adult cats, or even kittens with several weeks of *normal* visual experience, are almost all selective for a single orientation, and direction of stimulus movement, and have vigorous responses to these optimal stimuli (while maintaining a precise plotting of RF boundaries. As a group, these cells are usually binocular, but single cells occa-

sionally prefer one eye or the other⁵¹. The RFs for the two eyes are similar, but may be shifted relative to each other for detection of near or far images^{18,72,73}. The broad binocular facilitation seen in kitten neurons is replaced by narrow facilitation plus *suppression* at disparities neighboring the optimal depth.

Now the modeler knows the initial conditions and the normal end result of VC development. Next, consider what conditions influence the *time course* of normal and abnormal development.

First note that if VC neurons are to develop or maintain any selectivity, then the kitten requires *some* visual experience. Total visual deprivation (rearing in absolute darkness) results in virtually no orientation selectivity after the age of about eight weeks^{15,37,58,113}. It should be appreciated that even with dark rearing, the afferent axons to visual cortex have some *maintained* discharge. It is possible, with enucleation or tetrodotoxin, to eliminate this "non-visual" input, too^{38,55,105}.

Now let us list a number of *abnormal visual environments* which cause VC neurons to develop differently from normal.

- Temporary *monocular deprivation* leads to permanent ocular dominance (OD) shift, after which almost all cells give no response to the deprived eye¹¹².
- *Restricting the visual world to contours of a single orientation* skews the distribution of orientation preferences toward the experienced pattern^{10,23,48,49,81,104}.
- *Limiting vision to one direction of motion* biases VC cells to respond preferentially to that direction^{21,29,98,108}.
- Kittens raised viewing small dots develop cells with normal RF size, but preference for

unusually small spots within the RF^{79,110}.

- If kittens grow up in low temporal frequency strobe light, cortical neurons lose direction-selectivity^{26,22,27,74}.
- Using prisms to deviate the image in one eye relative to the other results in a compensatory shift in binocular receptive field disparity^{90,91}.

2. Outline of the Model

These results suggest the following theory.

At birth, each VC neuron is synaptically connected to enough afferent input so that a neuron can respond weakly to a great many stimulus arrangements. That subset of afferents which carries information during repeated presentation of stimuli finds its synapses strengthened, while the complement of that active subset finds its synapses weakened. By this use-disuse mechanism, the cell develops a response to only a selective set of stimulus patterns.

Modelling this sketchy, qualitative idea presents some difficulties. We need first to specify initial conditions, both anatomical and physiological, then to describe how a cell forms a single output from many inputs, then to express a rule by which inputs may have their influences changed, and finally, to show how this *dynamic* system with many cells can be *stable* over time.

3. Wiring Diagram for the Model

Four structures of the vertebrate CNS have been the favorite subjects of neuro-anatomists over the last century - retina, spinal cord, cerebellum, and neocortex⁸⁷. The first three of these have a limited number of cell types and a constrained set of connections between the cells. The neocortex, however, has a great diversity of cell types and a rich set of interconnections. See Figure 2. In considering how nerve cells of the retino-geniculate pathway are interconnected, the modeler has an embarrassment of riches.

For area 17, *visual* input comes principally from the lateral geniculate nucleus (LGN), with relatively minor inputs from the pulvinar and the claustrum⁸³. The dorsal LGN is divided into layers, physiologically defined by ocular dominance. Each layer contains cells excitable through one eye only. Inhibitory influences from the other eye can be demonstrated, however⁸⁵. Geniculate units show biases for the orientation of a moving slit^{25,112}. These biases, however, fall far short of the narrow tuning seen in cortical neurons. Modelers generally assume, therefore, that **the input to visual cortex has neither orientation selectivity nor binocularity.**

As documented by Mountcastle⁶⁶, and Hubel & Wiesel⁵¹, neocortex has a *columnar* organization. Cells recorded in a penetration perpendicular to the cortical surface have similar orientation preferences and ocular dominance (although cells outside of layer IV are generally binocular).

In addition to the *specific* afferents from thalamus, there are widespread *non-visual* inputs to VC: a noradrenergic projection from the locus coeruleus, a cholinergic from basal nucleus, and a serotonergic from raphe⁷¹.

The wiring scheme for VC models consists of excitatory afference from LGN combined with intracortical inhibition. Intracortical excitation has been added in some models for two main purposes: to tighten columnar organization, and to pass the signal up through the hierarchy. In order to model the columnar organization of cortex described above (see Figure 7), Malsburg⁶², Swindale¹⁰⁶, and others have studied networks with *short-range* excitatory intracortical connections and *longer-range* inhibitory interactions. Sillito⁹⁷ proposed a role for intracortical excitation in providing a baseline facilitation. Excitatory projections between cortical layers or areas can create reverberating circuits or hierarchical structures. Fukushima⁴⁰ has worked with abstract feature-extracting systems of several levels. Such networks, when plasticity is involved, present special problems^{5,84}. One way to deal with this complexity, is to utilize the technique of *supervised learning*. Assume the desired output is available to produce an error signal between the actual response and the goal, then synaptic modifications in a lower layer, based on the error signal from a higher layer provides the necessary control. Even so, sending error signals back over more than three layers can be costly, computationally.

Can we correlate cell types from anatomical study with components in our model circuit? In visual cortex, there are a few relationships established at the single-neuron level. Which cell types and synapses provide inhibitory and excitatory outputs can be reasonably guessed, but *response properties* cannot be predicted from morphology. One can assign inhibitory and excitatory model elements to smooth and spinous stellate cells respectively⁸⁶ or focus on basket cells as powerful inhibitory elements Daniels²⁸. Attempts to account for the role of each type of cortical cell remain speculative³¹.

It is the first task of the modeler to decide which aspects of this rich (and somewhat poorly understood) structure are essential for a successful wiring diagram of the model. Before solving this problem, however, the modeler must consider which connections are *excitatory*, which are *inhibitory*, and how to integrate these inputs to produce responses to visual stimuli.

4. Excitation and Inhibition

Excitation in VC comes via³¹

- the specific thalamic afferents,
- spiny stellate interneurons,
- collaterals of local pyramidal neurons,
- horizontal fibers in layer I,
- association fibers and commissural fibers emanating from distant cortical pyramidal cells,
- non-specific subcortical afferents⁷¹.

The majority of VC cells receive monosynaptic excitation from LGN, especially cells below layer II⁶⁵.

Inhibition, on the other hand, comes almost exclusively from local interneurons³¹ and from non-specific afferents. The *smooth* stellate cells are usually regarded as providing inhibitory outputs; these cells include basket cells, axoaxonic cells, axonal tuft cells, and double bouquet cells⁷⁵. GABA is probably the transmitter for these neurons⁸², and their synapses are located on either the initial segment and perikaryon, or the dendritic tree of pyramidal cells¹⁰². Two types of GABA receptors have been distinguished, and both localization and

function appear distinct for these receptors^{33,12,65}.

By excitation, we will generally mean monosynaptic afferent excitation; inhibition will always be polysynaptic from LGN. This intracortical inhibition is sometimes seen as simply an inversion of the sign of the afferent input (e.g. ref. 115), but it should be regarded as an interaction *between cortical cells* since inhibitory cells in VC have selective RFs⁶⁵, rather than aspecific LGN-type fields.

There is a spectrum of thought on the relative importance to VC specificity of excitatory and inhibitory mechanisms. Hubel and Wiesel⁵¹ relied on excitation to illustrate how their data on oriented RFs could be derived. Sillito⁹⁸ argued that inhibition generates these same RF properties. Between these extremes, various interactions between the two types of inputs have been proposed.

Figure 3 represents these alternatives in terms of tuning curves for the excitatory and inhibitory inputs to a cell. Models which emphasize excitation assume that selectivity arises through tuning the excitatory drive to the cell, with a relatively flat tuning curve for inhibition. Hubel and Wiesel⁵¹ suggested that the spatial response properties of VC cortical cells are built up hierarchically from excitatory convergence via non-selective LGN cells. They proposed that a first order orientation-selective cortical cell (simple cell) receives excitatory inputs from center-surround LGN cells whose centers were offset, but aligned along the long axis of the preferred orientation (Figure 4). Similarly, ocular dominance can result from the relative strength of the excitatory input of each eye. Hubel and Wiesel proposed that a set of simple cells preferring a common orientation excited a second class of neurons (complex cells), conferring their orientation selectivity while generalizing over a larger RF. Subsequent work⁵⁴ showed that, by and large, simple cells are stellate and complex cells pyramidal.

supporting Hubel and Wiesel's model.

Most models try to take this simple excitatory arrangement as far as possible before introducing inhibitory factors. Malsburg's model⁶² for development of orientation-sensitive cells features a hexagonal lattice of input elements. The elements are the centers of LGN receptive fields. His stimuli are bars which excited the central element in the lattice, and several elements on each side (Figure 5a). The cortical cells develop preferences for oriented stimuli by strengthening the connections from a set of aligned elements, resulting in the arrangement suggested by Hubel & Wiesel (Figure 5b).

Bienenstock et al.⁷, started with inputs generated by bars overlapping elements on a lattice (Figure 6). Strong excitatory connections from *one* of these oriented inputs come to dominate a VC cell, thereby determining the cell's optimal orientation.

Each of these models follows the pattern of figure 3a, where the excitatory connections are organized essentially in the form of the preferred input. Each model also implements an additional input which enhanced selectivity to a relatively minor degree, through inhibition of non-optimal patterns.

Malsburg combined intracortical interactions of *short-range* excitation and *longer-range* inhibition (see Figure 7). This led to a spatial organization of the cortical layers in which neighboring cells developed preference for *similar* orientations. Malsburg's long-range inhibition brought dissimilar patterns into opposition. Suppression of nonoptimal patterns through inhibition was also used by Bienenstock et al.⁷ to sharpen orientation selectivity. Excitatory convergence produced the basic orientation tuning while inhibition suppressed remnant responses to non-preferred patterns, essentially by raising the threshold. This overall

approach implies that excitation is the primary carrier of selectivity information, and that inhibition is used secondarily to improve tuning, and perhaps add a stabilizing influence.

Others have considered the opposite relationship between excitation and inhibition: that most of the selectivity is achieved by *inhibition of all but the optimal pattern*, and that excitation merely provides a *background* to be inhibited (Figure 3c). Creutzfeldt et al.¹⁹ pointed out that an orientation-sensitive cortical cell might receive excitation from a *single* LGN afferent^{47,57,95}, and if so, then it should show no selectivity were it not for the influence of an *asymmetrical* intracortical inhibition. Heggelund & Moors⁴⁶ proposed that oriented RF's result from two circularly symmetric inputs whose centers are slightly offset, one of which is inhibitory and the other excitatory (Figure 8a). Braitenberg¹³ extended that arrangement to produce a *two-dimensional* sheet of orientation-selective cells. Sillito⁹⁷ proposed further elaborations of Heggelund's scheme (Figure 8b) using several stages of inhibition. Barlow & Levick⁴ and Hassenstein & Reichardt⁴⁴ provided *experimental* evidence that direction-selectivity in *retinal* neurons is due to asymmetric inhibition. Cortical direction selectivity depends similarly on inhibition^{33,48,55,97}. DeValois & Tootell³⁰ noted an asymmetric inhibition in spatial frequency tuning, finding inhibition from *harmonics* of the best frequency. Furthermore, Hubel and Wiesel's strict hierarchical model of excitatory convergence from LGN to simple cells to complex cells has been called into question by a number of results showing, for example, that complex cells receive monosynaptic input from LGN^{52,67} and respond to noise stimuli which fail to excite simple cells⁴⁴. The evidence for a complete correlation between the stellate/pyramidal and simple/complex classifications has also been weakened^{42,63,68}.

Sillito has argued most strenuously for the alternative illustrated in Figure 3c. He observed the excitatory tuning curve after applying iontophoretically the GABA antagonist

bicuculline⁹⁶⁻¹⁰¹. During bicuculline application, normally sharply tuned cells lose all selectivity⁹⁵. Based on his own data, as well as other evidence, Sillito concluded that the excitatory input from LGN has a flat tuning curve, while the intracortical inhibition is highly organized. He proposed that inhibition "...is optimally effective at orientations away from the preferred optimum and hence serves to generate selectivity to that optimum" (101, page 109). Sillito places *intracortical* (not afferent) *excitation* as an important but secondary agent in generating RF properties.

We suggest that *both* excitation and inhibition contribute to response properties. In this compromise view, *both excitation and inhibition are tuned around a common optimum* (Figure 3b). Blakemore and Tobin⁸ found examples of VC neurons which were inhibited most effectively by gratings parallel to the cells' preferred orientations. From *intracellular* recordings, Ferster³⁵ produced orientation tuning curves of the type in Figure 3b. Allman et al.² have recently reviewed the common occurrence of extensive input from outside the classical excitatory RF, and discuss the evidence that *inhibition is tuned to match the response properties of VC cells*.

This compromise alternative can support a range of possibilities: it could allow excitation to be broadly tuned, since broadly tuned inhibition would effectively eliminate responses to non-optimal stimuli. On the other hand, excitation and inhibition could both show sharp tuning. Either way, the difference between this compromise situation (Figure 3b) as opposed to the extremes (Figure 3a or 3c) is that here, excitation and inhibition are tuned in concert with each other and with the resultant response.

Different response dimensions undoubtedly draw on varying mixtures of excitation and inhibition. Ocular dominance probably depends primarily on the balance of excitatory inputs

from the two eyes, whereas directionality and binocular disparity probably require inhibition to generate sharp tuning; orientation, spatial position, and spatial frequency specificity probably benefit from equal combinations of inhibition and excitation.

5. Synaptic Modification Rules

Once a reasonable wiring diagram is in place, and connections are labelled as excitatory or inhibitory, a rule for changing connection *strengths* is needed, in order to see simulated development. In some cases, the initial wiring is assumed to have excess connections, resulting in little specificity. Synaptic modification then eliminates certain connections. Sometimes a mild topological constraint is imposed on the diffuse initial state: cells, including afferent axons, make synapses with *neighboring* cells, rather than with the entire network. An initial *bias* in the wiring is necessary in Malsburg's systems^{65,66} because a seed for further organization is needed. *Fluctuations from noisy input* are sufficient to break the initial symmetry and achieve highly asymmetric final states in Bienenstock et. al⁷.

Various combinations of *modifiable and non-modifiable synapses* have been employed. Each of the alternatives discussed above and illustrated in Figure 3 corresponds to a decision about how to modify excitatory and inhibitory synapses. Both Malsburg⁶⁵ and Scofield⁹⁰ used fixed intracortical synapses and modified only the thalamocortical excitatory synapses. Their results are of the type in Figure 3a. Nagano & Kurata⁷³ modeled the development of complex cells using fixed excitatory synapses from LGN and modifiable inhibitory connections from simple cells, giving the sort of inputs described by Figure 3c. The natural assumption to make to obtain the results of Figure 3c, or even Figure 3a, is that active inhibitory synapses are weakened and inactive inhibitory synapses are strengthened. This assumption

facilitates responses to experienced stimuli^{7,62,70}. In order to arrive at the inhibitory tuning curve of Figure 3b, one can assume that *inhibitory synapses that are active are strengthened*, just as for excitatory synapses. Wilson¹¹⁸ modified intracortical inhibitory synapses in this manner, although in his "anatomy", he separated the inhibitory cortical cells from the output cells, essentially reducing the inhibition to a "retinal afference". Saul⁸⁹ modified both excitatory afference and true intracortical inhibition, showing how VC cells can develop specificity dependent on both excitation and inhibition.

Rules for synaptic modification span a broad range; see Table I. Hebb⁴⁶ postulated that learning involved an increase in the synaptic efficacy between two cells which were simultaneously active. His rule encodes into the synapse the correlation between pre- and postsynaptic activity [eqn A of Table I]. Many others have started with Hebb's rule to create different modifiable synapses.

Stent¹⁰⁶ not only suggested the applicability of Hebbian modification to the visual system, but also proposed a specific (though speculative) physiological mechanism: The "ejection" of neurotransmitter receptors from the postsynaptic membrane during the *polarity reversals* which accompany cell firing, *unless* local synaptic activity stabilizes the receptors by holding the membrane potential below zero. In Stent's model, activity in the postsynaptic cell during *inactivity* of the presynaptic terminal results in *decreasing* the strength of the synapse.

In order to *increase* the strength of a synapse, the rule can uniformly strengthen (with low gain) *all* synapses on the postsynaptic cell. In this case, inactive synapses lose a lot, but add only a little, while active synapses strengthen slightly. By this means, the modification of Figure 9a and equation B is obtained. This scheme implies that no change occurs in the

absence of postsynaptic firing, and is sometimes referred to as *postsynaptic resonance*⁸³.

Stent's mechanism could also be applied to inhibitory synapses (since ipsp's would certainly prevent local polarity reversals), where the modifications turn out to be identical to those for excitatory synapses (as in 32, 89, 118; see eqns C, D, M). This generates the tuning curve in Figure 3b.

Von der Malsburg⁶⁵ modified his excitatory afferent synapses according to Hebb's rule. Concurrent activity in the i th LGN cell ($x_i \neq 0$) and the k th cortical cell ($y_k \neq 0$) induces a change in the connection strength $\Delta s_{ik} = h x_i y_k$. To be able to *decrease* strengths, he normalized the connections onto a given cortical cell to result in a constant sum of all their strengths. Von der Malsburg therefore obtained the modification "table" of Figure 9a (and equation E), as in the Stent-type model. Perez et al.⁸⁰ followed a similar procedure (equation F), strengthening by the Hebb rule, followed by weakening due to conservation of total synaptic strength.

Amari & Takeuchi³ also follow Hebb, but build an *exponential decay term* into their modification rule (Figure 9b and eqn G of the Table). In this scheme, strong synapses always decay, setting their level at the product of pre- and postsynaptic activity. Such a rule is more amenable to analysis than others, which *must be simulated*. (Ullman & Schechtman¹¹³ and Easton & Gordon³² use similar rules for ease of analysis, equations K, L, and M.) They also proposed a variant with the property of postsynaptic resonance, shown in Figure 9a and eqn H. Identical rules were applied to excitatory and inhibitory synapses. They used a *single* inhibitory input of given amplitude, so their inhibitory modifications simply provided a threshold-setting parameter to separate categories of patterns coming in through the excita-

tory inputs.

In their model for the development of directional selectivity, Nagano & Fujiwara⁷² assumed that active inhibitory synapses *weakened* as in Figure 9c and eqn I. Their model produces directional preference in the absence of other features, and it is able to reproduce visual deprivation-experiment results in which either all cells preferred a single experienced direction or no cells showed directionality. They assume that a cortical cell completely inhibits (vetoes) its neighbors. The modification rule permits movement in the *preferred* direction to eliminate the inhibitory block, which remains for movement in the *null* direction (as in Figure 3c). Note that this modification scheme is explicitly time-dependent. *Any of the other above equations can be made to depend similarly on delays between pre- and post-synaptic firing.*

Bienenstock et al.⁷ used a more complicated learning rule. They replaced the postsynaptic factor in the simple Hebbian product (eqn A) by a *nonlinear* function of both the postsynaptic firing and a *modification threshold* (which is in turn a function of past postsynaptic activity, eqn J). The form of this function weakens synapses when postsynaptic response is small and strengthens them when responses are large relative to the modification threshold. The modification table is shown in Figure 9d, although the actual behavior of equation J is much richer than this caricature since the modification threshold adapts to the cell's activity, as will be discussed below. The modification rule does not assume postsynaptic resonance, but it has a presynaptic resonance built in, as well as an implied need for some postsynaptic activity to *raise* the modification threshold. Bienenstock et al.⁷ applied this rule to a *bipolar* synapse which could range through negative and positive influences, which means that the modification could *reverse sign* as in Figure 9e when the synapse passed from excitatory to

inhibitory. Scofield⁹⁰ separated the bipolar synapse into excitatory and inhibitory parts and chose to modify only excitatory synapses, applying the same rule (equation J). Analysis shows that the two key reasons this rule reproduces some of the classic deprivation results are (1) the *competition* induced by the positive and negative regions of the ϕ -function and (2) the stabilization produced by pushing the positive region ahead of the average activity of the cell. This will be discussed in detail in the next section.

Non-Hebbian rules have also been applied to visual cortical modeling. Lara & Di Prisco⁵⁹ modeled interocular competition during development by assuming that pre-synaptic processes such as post-tetanic potentiation and heterosynaptic inhibition and sensitization have prime importance. Although they included a *regulatory* influence from the post-synaptic activity, *their mechanisms were not related to correlations between pre- and postsynaptic elements, but depended on competition and cooperation only between pre-synaptic fibers.* Swindale¹¹⁰ and Malsburg⁶⁶ obtained similar results for the development of *ocular dominance stripes*, without resorting to such a detailed mechanism.

6. Stability of the Selective State

There are a number of different *stability* problems inherent in developmental specificity models. The key feature of almost all these models, however, is that *states which represent selectivity are stable.* In anthropocentric terms, selectivity means that a cell "decides" whether to fire based on "knowledge" of which particular inputs are firing, and can therefore "make decisions" about changing its state. In vector terms, most modification rules move the synaptic state (the vector of synapses) parallel to some input vector until the state nears the equilibrium point corresponding to specificity for that stimulus. As this steady state is

approached, the modification rule becomes dominated by the decay toward the selective state. Amari & Takeuchi³ provided an elegant demonstration of this result. The steady state of equation G is the average over the input patterns weighted by the cell's responses to the patterns. Visual experience gives synapses a chance to find this state by **averaging over time**. The decay term ensures that any transient perturbations are attenuated.

Hebb's postulate is often seen as permitting only *increases* in synaptic efficacy in response to positively correlated activity. Such increases cannot continue unbounded.

Malsburg⁶⁵ and Perez et al.⁸⁰ simply *normalized* their synaptic strengths to conserve the total strength onto a cell. This normalization provides both a decremending process and a bound on synaptic strength. Their normalization is *instantaneous*, but the desired results can be obtained more realistically by combining a fast Hebbian process with a slow asymptotic approach toward a constant sum (eqn B). The physiological bases for conservation rules are finite availability of transmitter and limited number of post-synaptic receptors. Because of enzyme kinetics, it seems reasonable to postulate a slow change in transmitter and receptor distributions. When placed in a model, these slow processes can produce an apparent *consolidation period* between experience itself and the final synaptic strengths wrought by that experience [For experimental support, see 16, 79; but see 37 for the opposite result]. In Wilson's system¹¹⁸, a synaptic conservation hypothesis helped account for psychophysical data on adaptation to low spatial frequencies. In another point of view, Grossberg⁴³ has argued that a simple shunting-type adaptation can replace conservation rules, by which he meant simply that the conservation rule can be shifted to the presynaptic layer, since strong inputs from one presynaptic cell will reduce the activity in other presynaptic cells using lateral inhibition.

Cooper's model⁷ relies on the *postsynaptic* activity in order to achieve stability. The form of the ϕ -function in eqn J is chosen so that the synaptic weights converge to appropriate values as long as the input environment is stable (in the sense of being a *stationary stochastic process*-- c.f. ref. 6). The essence of the ϕ -function's role in stability is that the modification threshold, which separates the weakening and strengthening zones, adapts to the cell's activity in the following manner: Θ_M eventually rises above all responses except the response to a single optimal pattern. This optimal pattern produces improved responses because the associated ϕ is positive, whereas ϕ is negative for other patterns, leading to reduced responsiveness to those non-optimal patterns. Once these non-preferred patterns come to produce no response at all, the modification threshold catches up to the optimal pattern response so that ϕ is zero for all patterns. The negative slope of ϕ at zero response stabilizes the non-optimal patterns, while the potentially unstable positive slope at the preferred pattern's steady-state response finds the modification threshold varying *more quickly* in the same direction as the response. At the steady state, this adaptation effect dominates, pushing the ϕ -function back toward zero (Figure 10).

Suppose a model moves to a stable state during exposure to a particular environment. What will happen if the environment changes? Consider the "reverse-suturing" paradigm where a formerly closed eye is opened, and the other eye closed, while still in the critical period. An OD shift toward the last-opened eye is seen^{9,70} (Figure 11). We can regard the reversal as a *breakdown of stability*. We then might presume that the lack of reversal at later ages is due to increased stability after longer exposure to the original open-eye environment. This increasing stability accompanies the formation of the adult state (extreme monocularity in the reverse-suture example) in many models of cortical development. However, steady-

environment stability is often unable to handle drastic change in the stimulus environment, and in models such as Bienenstock et al.⁷ there is nothing to stop another complete shift. Instead of relying on intrinsic stability therefore, one may have to use an *outside gating* of plasticity, as has been suggested by various experiments (17, 36, 53, 103, 104). Detailed models for external gating have not been published, but we should expect that signals derived from the state of visual cortex should inform the gating mechanism so that plasticity is reduced at the appropriate time, i.e. when specificity has been achieved. In what form this global regulator of plasticity arrives at the local visual cortical mechanisms is not clear, although this is an active area of research⁶¹. The next generation of modeling efforts must include global control mechanisms to modulate local synaptic strengths.

Summary

We review models for development of selectivity in kitten visual cortex neurons. The models rely on an understanding of the *afferent connections* to VC, and on the *intrinsic connections* between layers in cortical columns. *Excitatory and inhibitory labels* are placed on the connections in the model's structure. Modification rules for some of the synapses allow the model systems to change (develop) in response to a visual environment. The changes stabilize on states which represent *selectivity* in single-unit responses, provided the visual environment itself has not changed greatly during the development.

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Table I -- Modification Equations

x_i = i-th retinal/geniculate cell activity

y_k = k-th cortical cell activity

s_{ik} = weight of excitatory connection from cell i to cell k

q_{lk} = weight of inhibitory connection from cell l to cell k

$$\dot{s} = ds/dt, \Delta s = s_n - s_{n-1}$$

A) Hebb⁴⁶ (excitatory): $\Delta s_{ik} = h x_i y_k$,

h is a time constant giving learning rate.

B) Saul⁸⁹ (excitatory): $\dot{s}_{ik} = -h_s (1-x_i) y_k s_{ik} + \frac{c_s}{N} (s_{ok} - \sum_{j=1}^N s_{jk})$,

h_s and c_s are time constants, N is the number of retinal cells projecting to cortical cell k , s_{ok} is the constant sum of synaptic strength onto cell k , and $0 \leq x_i, y_k \leq 1$.

C) Saul⁸⁹ (inhibitory): $\dot{q}_{lk} = -h_q (1-y_l) y_k q_{lk} + \frac{c_q}{M} (q_{ok} - \sum_{h=1}^M q_{hk})$,

analogous to B) but for intracortical inhibition.

D) Wilson¹¹⁸ (inhibitory): $\Delta q_{lk}(t) = \lambda \int_0^t h(t-\tau) y_l(\tau) y_k(\tau) d\tau$.

the kernel h monotonically decreases toward zero: $h(t) = e^{-t}$, λ a constant.

E) Malsburg⁶⁵ (excitatory): $\Delta s_{ik} = \alpha h x_i y_k - (1-\alpha) s_{ik}$, h is a time constant,

$$\alpha = s_{ok} / \sum_{j=1}^N (s_{jk} + h x_j y_k),$$

s_{ok} is the constant sum as in B.

F) Perez et al⁸⁰ (excitatory): $\dot{s}_{ik} = hx_i y_k - \gamma (1-x_i) s_{ik}$,

$$\gamma = h \sum_{j=1}^N x_j y_k / \sum_{j=1}^N (1-x_j) s_{jk}.$$

G) Amari and Takeuchi³ (excitatory): $\dot{s}_{ik} = -\alpha s_{ik} + hx_i y_k$,

α, h are time constants, $y_k \in \{0, 1\}$.

H) Amari and Takeuchi³ (excitatory): $\dot{s}_{ik} = (-\alpha s_{ik} + hx_i) y_k$,

α, h as in G.

I) Nagano and Fujiwara⁷² (inhibitory): $\Delta q_{ik}(t) = (\alpha - hx_i(t-2)) y_k(t)$,

x_i and y_k functions of (discrete) time, α, h positive time constants, $0 < x_{\min} \leq x_i \leq x_{\max}$ when cell i is excited, $x_i = 0$ otherwise, and $\alpha - hx_{\min} < 0$.

J) Bienenstock et al.⁷ (excitatory and inhibitory): $\dot{s}_{ik} = \phi(y_k, \Theta_M) x_i$,

ϕ a particular function of y_k and Θ_M with Θ_M being a modification threshold which is a function of \bar{y}_k , a running time average of y_k -- see Figure 10.

K) Ullman and Schechtman¹¹³ (excitatory): $\Delta s_{ik} = h(\alpha - s_{ik} x_i) = h(\alpha - y_k)$,

α a constant norm, h a time constant.

L) Easton and Gordon³² (excitatory): $\dot{s}_{ik} = h_s (x_i y_k - s_{ik})$,

h_s a time constant.

M) Easton and Gordon³² (inhibitory): $\dot{q}_{ik} = h_q (y_k^2 - q_{ik})$,

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h_q a time constant.

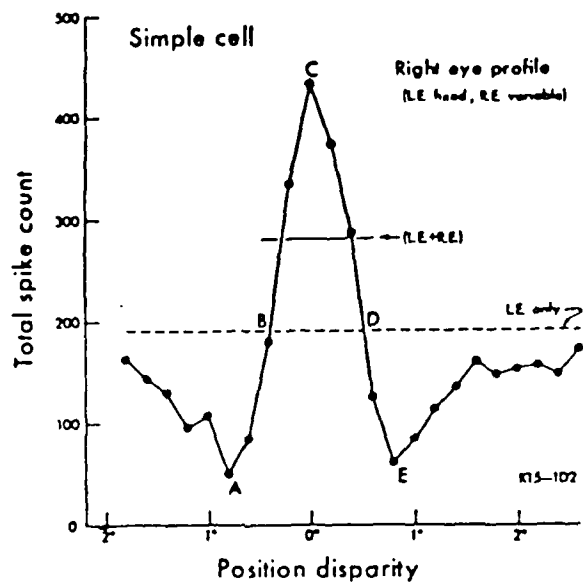


Figure 1. Binocular disparity tuning curve from a cat VC simple cell, from Nelson et al. 1977, reproduced with permission. This cell is sharply tuned, responding over about 1° of retinal disparity. The 0° reference is somewhat arbitrary. Misalignment of the two retinal images from their optimal positions leads to suppression of the response below the level of the monocular responses. The dashed line shows the response to the left eye stimulus alone, which was fixed for this curve. The right eye stimulus was advanced or delayed from its optimal position. The short continuous line shows the sum of the monocular responses at the optimal positions. Binocular stimulation thus produces facilitation over a narrow range of disparities.

Table 1. Modification Equations

- x_i = i -th retinal/geniculate cell activity
 y_k = k -th cortical cell activity
 s_{ik} = weight of excitatory connection from cell i to cell k
 q_{ik} = weight of inhibitory connection from cell i to cell k
 $\dot{s} = ds/dt$, $\Delta s = s_{\text{new}} - s_{\text{old}}$
- A. Hebb¹⁰ (excitatory): $\Delta s_{ik} = hx_i y_k$.
 h is a time constant giving learning rate
- B. Saul¹¹ (excitatory): $\dot{s}_{ik} = -h(1-x_i)y_k s_{ik} + \frac{c}{N}(s_{ik} - \sum_{i=1}^N s_{ik})$.
 h and c are time constants. N is the number of retinal cells projecting to cortical cell k . s_{ik} is the constant sum of synaptic strength onto cell k , and $0 \leq x_i, y_k \leq 1$.
- C. Saul¹¹ (inhibitory): $\dot{q}_{ik} = -h(1-y_k)q_{ik} + \frac{c}{M}(q_{ik} - \sum_{i=1}^M q_{ik})$.
 analogous to B. but for intracortical inhibition.
- D. Wilson¹² (inhibitory): $\Delta q_{ik}(t) = \lambda \int_0^t h(t-\tau)y_i(\tau)y_k(\tau)d\tau$.
 the kernel h monotonically decreases toward zero: $h(t) = e^{-t}$, λ a constant.
- E. Malsburg¹³ (excitatory): $\Delta s_{ik} = \alpha h x_i y_k - (1-\alpha)s_{ik}$, h is a time constant.
 $\alpha = s_{ik} / \sum_{i=1}^N (s_{ik} + h x_i y_k)$.
 s_{ik} is the constant sum as in B.
- F. Perez et al.¹⁴ (excitatory): $\dot{s}_{ik} = h x_i y_k - \gamma(1-x_i)s_{ik}$.
 $\gamma = h \sum_{i=1}^N x_i y_k / \sum_{i=1}^N (1-x_i)s_{ik}$.
- G. Amari and Takeuchi¹ (excitatory): $\dot{s}_{ik} = -\alpha s_{ik} + h x_i y_k$.
 α, h are time constants, $y_k \in \{0, 1\}$.
- H. Amari and Takeuchi¹ (excitatory): $\dot{s}_{ik} = (-\alpha s_{ik} + h x_i y_k)y_k$.
 α, h as in G.
- I. Nagano and Fujiwara²³ (inhibitory): $\Delta q_{ik}(t) = (\alpha - h x_i(t-2))y_i(t)$.
 x_i and y_i functions of (discrete) time, α, h positive time constants, $0 < x_{\min} \leq x_i \leq x_{\max}$ when cell i is excited, $x_i = 0$ otherwise, and $\alpha - h x_{\min} < 0$.
- J. Bienenstock et al.⁷ (excitatory and inhibitory): $\dot{s}_{ik} = \phi(y_i, t) x_i$.
 ϕ a particular function of y_i and t , with t_0 being a modification threshold which is a function of \bar{y}_i , a running time average of y_i —see Figure 10.
- K. Ullman and Schechtman¹¹¹ (excitatory): $\Delta s_{ik} = h(\alpha - s_{ik}) = h(\alpha - y_k)$.
 α a constant norm, h a time constant.
- L. Easton and Gordon¹² (excitatory): $\dot{s}_{ik} = h(x_i y_k - s_{ik})$.
 h , a time constant.
- M. Easton and Gordon¹² (inhibitory): $\dot{q}_{ik} = h(y_k^2 - q_{ik})$.
 h , a time constant.

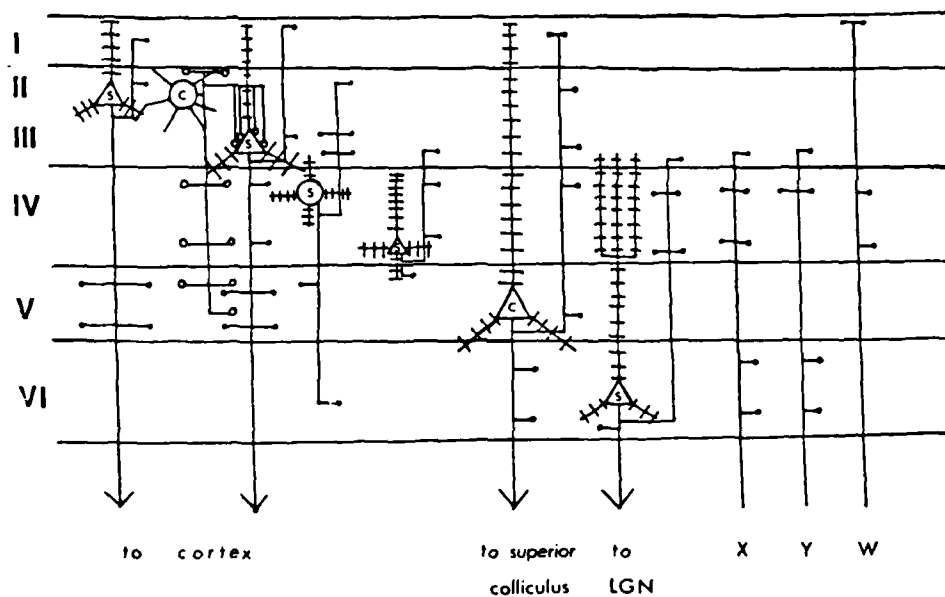


Figure 2. Schematic diagram of cortical organization. Specific inputs arrive via the X, Y, and W pathways from LGN. Layer IV contains stellate cells and some small pyramidal cells with stellate-like dendritic arborizations. Simple type receptive fields (S) predominate. Other layers contain predominantly pyramidal cells. Their fields can be simple or complex (C). Smooth stellate cells can be found throughout cortex, however, and one of these is illustrated in layer II as a *basket cell*. Outputs from superficial layers project to other cortical locations, while layers V and VI project to superior colliculus and LGN respectively.

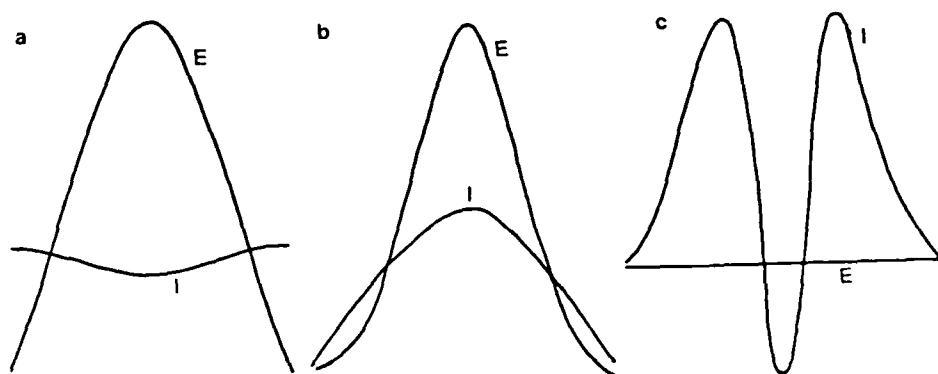


Figure 3. A simplified view of the possibilities for tuning of excitatory and inhibitory inputs to a cell. (A) Excitation is tuned and generates the final response tuning with little contribution from inhibition. The inhibitory input may be strong, but it is relatively flat. Inhibition may be reduced at the optimum in order to facilitate response. (B) Both excitation and inhibition are tuned in concert with the response: they share a common optimum. Both types of inputs contribute to the final response tuning. (C) Inhibition is highly tuned while excitation is flat. The inhibition serves to eliminate response to nonoptimal stimuli while not blocking optimal stimuli. The excitation provides only a background drive.

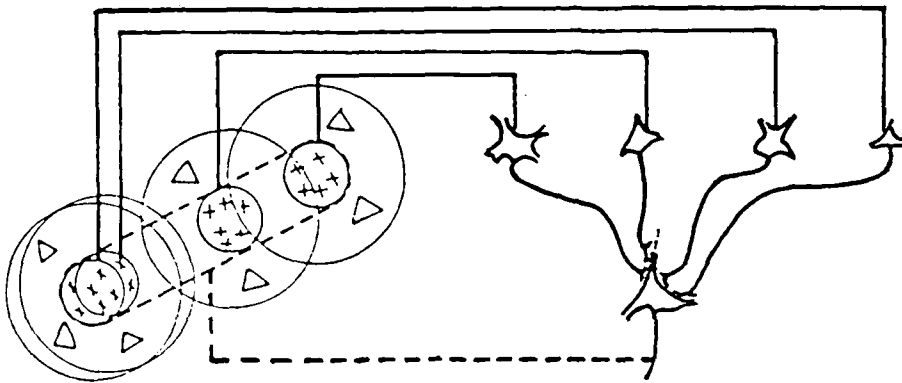


Figure 4. Hypothetical construction of an oriented VC cell RF, from Hubel & Wiesel 1962, reproduced with permission. A large number of LGN cells, including the four cells with the ON-center/OFF-surround fields at the left, project onto the cortical cell at the bottom right. This cortical cell would, therefore, be endowed with the RF indicated by the dashed outline.

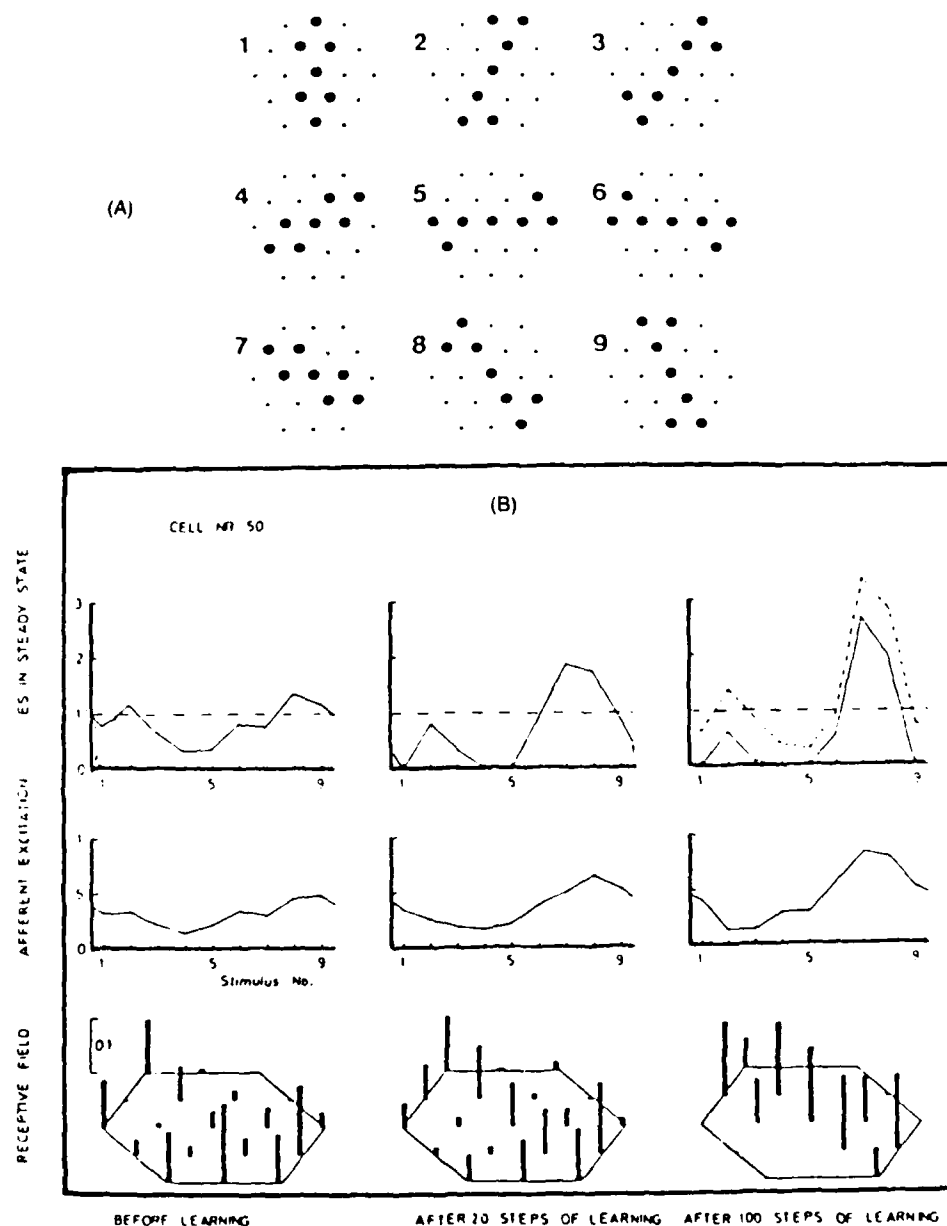


Figure 5. Von der Malsburg (1973) modeled the development of oriented RFs by assuming an excitatory projection from *retina* to *cortex*. (A) The 19 retinal cells were arranged in a hexagonal array, and nine standard stimuli were formed as indicated by the large or small dots which corresponded to active or inactive retinal cells. (B) The response of one of the cortical cells is shown. At the top, the responses to each of the nine standard stimuli are drawn for three points in time: before learning, after 20 learning trials, and after 100 learning trials took place. Initially, the cell was relatively aspecific (though biased), but by the end had become selective for patterns #7 and #8. The dashed line shows the firing threshold, and the dashed curve on the right shows the response after removal of inhibition. The second row gives the net afferent input for each pattern. In the third row, the heights of the bars represent the synaptic strengths from each retinal fiber onto the cell. Synapses which are active during presentations of patterns #7 and #8 are the only remaining connections. Reproduced with permission.

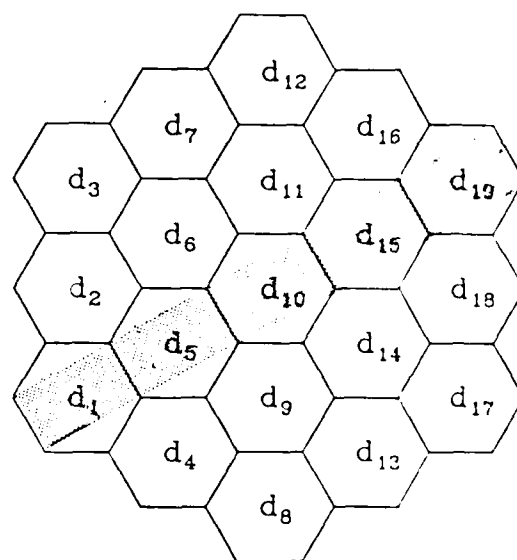


Figure 6. The pattern generator used in simulations of Cooper's model consisted of 19 retinal cells. A bar was projected on this array, and the area of each cell which was covered by the bar provided a value for the activity in that cell.

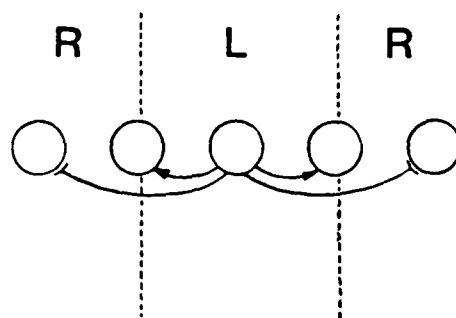


Figure 7. An often-assumed intracortical wiring scheme is short-range excitation (—>) and longer-range inhibition (—|). These connections can be used as a force to produce columnar organization, with cells in a column providing mutual excitation and cells in neighboring columns providing mutual inhibition.

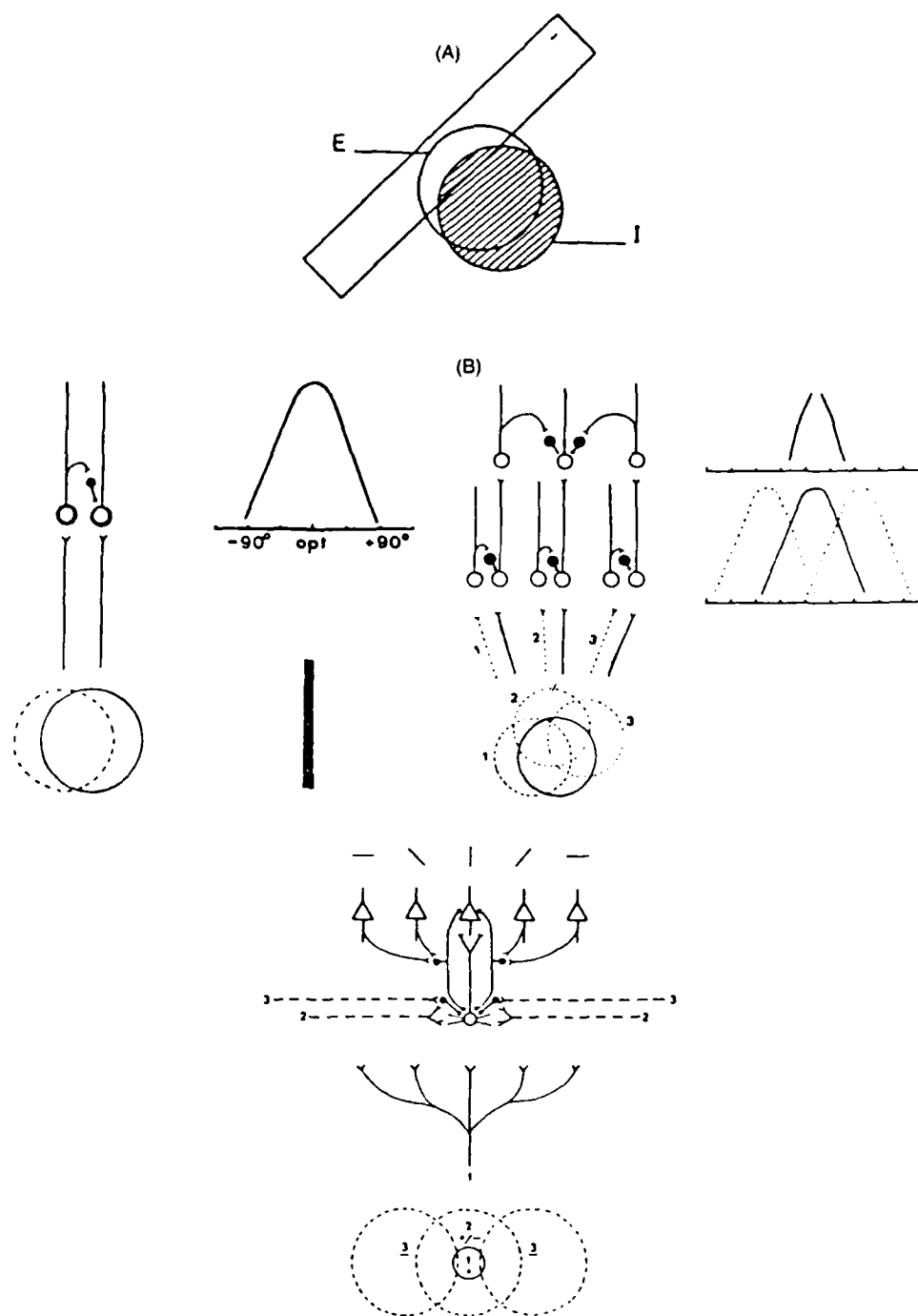


Figure 8. (A) Heggelund and Moors (1984) hypothesized that orientation selectivity arises from overlapping subfields of opposite effect. Each subfield is circular, but the partial overlap leaves an elongated excitatory zone exposed. Reproduced with permission. (B) A more complex diagram from Sillito (1985) which uses the scheme in A (upper left). In the upper right, three of these channels interact to produce a secondary field with enhanced selectivity. At the bottom, the inhibitory fields are large relative to the excitatory field ("1") and field "2" is mixed inhibitory and facilitatory. The inhibitory interactions now include several types of lateral projections, along with a feedback from the secondary units (triangles) to the primary cells (circle in middle). Reproduced with permission.

a	PRE \ POST	OFF	ON
		OFF	ON
	OFF	0	—
	ON	0	+

b	PRE \ POST	OFF	ON
		OFF	ON
	OFF	—	—
	ON	—	+

c	PRE \ POST	OFF	ON
		OFF	ON
	OFF	0	+
	ON	0	—

d	PRE \ POST	LOW	HIGH
		LOW	HIGH
	LOW	0	0
	HIGH	—	+

e	PRE \ POST	LOW	HIGH
		LOW	HIGH
	LOW	0	0
	HIGH	+	—

Figure 9. Modification tables for various Hebb-type rules. See text for details. PRE = presynaptic activity, POST = postsynaptic activity. The tables show the sign of the synaptic change: + = increase strength, — = decrease strength, 0 = relatively little change. c and e are for inhibitory synapses, where anti-Hebbian rules have sometimes been applied, which accounts for the — sign in the bottom right quadrant, rather than the Hebbian + sign. 0s in the first column indicate *postsynaptic resonance*, in which postsynaptic activity is required to gate modifications.

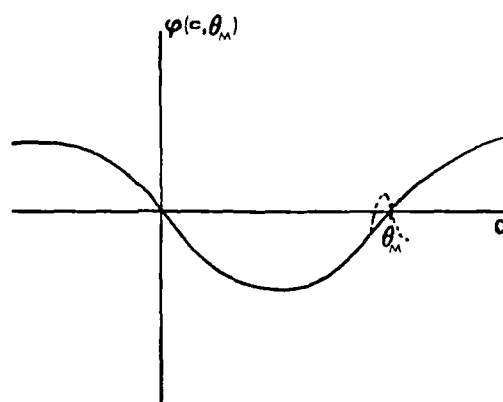


Figure 10. The ϕ -function of equation J, after Bienenstock et al. 1982. ϕ is a function of postsynaptic activity and a modification threshold θ_M . For fixed θ_M , ϕ is positive for $y < 0$, ϕ is negative for $0 < y < \theta_M$, and ϕ is positive for $y > \theta_M$ (see Figure 9d). However, θ_M is a function of the average postsynaptic activity, which becomes very important at the steady-state, when $y \approx \theta_M$. Under appropriate conditions, this nonlinear dependence of θ_M on y effectively curves the ϕ -function back toward zero locally around the equilibrium point (dashed curve).

Models for Visual Cortex

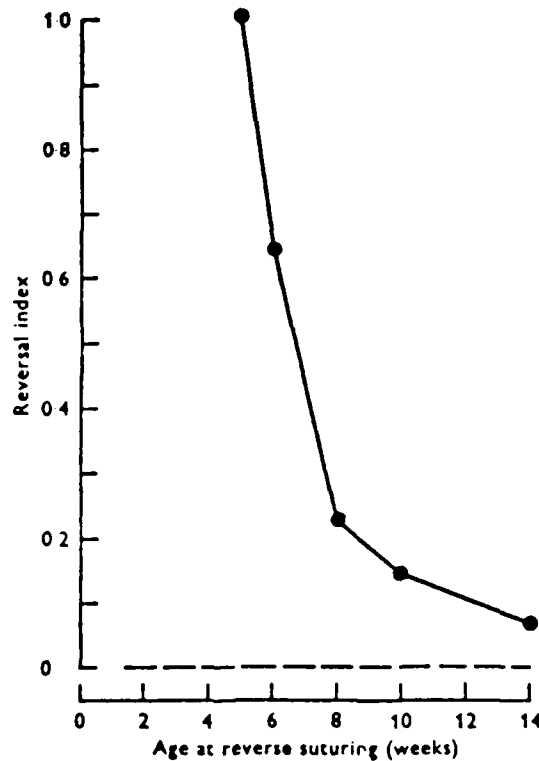


Figure 11. Increasing resistance to ocular dominance shift toward the last-opened eye after reverse-suture at later ages, from Blakemore and Van Sluyters 1974, reproduced with permission. The reversal index is the proportion of cells dominated by the last-opened eye. The dashed line at 0.0 is the reversal index for a control animal, which was initially deprived until 5 weeks, but not reversed-sutured. These data show that plasticity decreases and stability increases during the critical period. This stability could depend solely on age, but it may also rely on visual experience, since the older reverse-sutured subjects had longer initial deprivations.

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